THYROID FUNCTION IN THE EARLY PERIOD AFTER RESUSCITATION

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UDC 616-036.882-08-07:616.441-008-07

The dynamics of the content of protein-bound iodine (PBI) in the early period after resuscitation from clinical death lasting 5 min caused by blood loss and interruption of the circulation for 10-12 min produced by electric shock was investigated in experiments on 33 dogs. The character of changes in thyroid function after the terminal state was closely connected with the eventual result of resuscitation. In cases when a decrease in the PBI content of the plasma was observed 6 h after resuscitation, the animals showed restoration of CNS function. In animals which die 12-24 h after resuscitation, a marked increase in the PBI concentration in the blood was observed.

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The results of resuscitation after clinical death are closely related to the character of responses of the endocrine system in the posthypoxic period. This has been demonstrated, in particular, for the pituitary–adrenal cortex system [1].

The object of this investigation was to study the dynamics of the content of thyroid hormones in the plasma in the recovery period after various types of terminal state.

EXPERIMENTAL METHOD AND RESULTS

Two series of experiments were carried out on 33 dogs. In the experiments of series I (20 animals) clinical death for 5 min was produced by acute blood loss. In series II (13 animals) the circulation was stopped for 10-12 min by electric shock. The animals were resuscitated by the method of Negovskii and co-workers [6]. The protein-bound iodine (PBI) of the plasma was determined by a modified Barker's method [7]. Blood for investigation was taken from the animals' femoral artery in the initial state, during agony, after restoration of the corneal reflexes, and 6, 12, and 24 h after resuscitation.

Resumption of cardiac activity in most animals in the experiments of series I took place 0.5-1.3 min after the beginning of resuscitation, and as late as 1.5-5 min after resuscitation only if fibrillation of the heart developed (6 experiments). After resuscitation of the animals in series II the arterial pressure was maintained for 2-3.7 min by external cardiac massage at the level of 60-100 mm Hg, after which the cardiac activity could be restored by electrical defibrillation. Respiration and the corneal reflexes recovered (Table 1) more rapidly in animals surviving after clinical death.

The character of changes in the plasma PBI concentration in postterminal states varied depending on the outcome of resuscitation (Table 2). In animals surviving clinical death and subsequently regaining the functions of their central nervous system, the plasma PBI level was lowered in the posthypoxic period. If the recovery period followed an unfavorable course, the plasma PBI concentration was increased 6 h after clinical death. These animals died as a rule between 12 and 24 h after resususcitation. The plasma PBI level 6 h after resuscitation was 3-4 times higher in animals which subsequently died than in those which survived.

When the postresuscitation period followed a favorable course, depression of the secretory function of the thyroid was thus depressed, and thyroid hormones were utilized by the tissues, but if the neurological state of the animals after resuscitation was poor, thyroid function was activated and thyroid hormones accumulated in the blood. The results of these experiments are in agreement with data in the literature

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Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 69, No. 2, pp. 23-25, February, 1970. Original article submitted June 6, 1969.

TABLE 1. Character of Development of Terminal State and Resuscitation after Clinical Death (M±m)

Series of experiments	Time taken to produce terminal state (in min)	Recovery (in min)		Outcome of resuscitation	No. of animals
		of respiration	of corneal reflexes		
I	12,51±1,45	4,36±0,36	12,24±0,85	Survival	11
	14,26±1,92	8,51±1,31 $P<0,01$	16,99±2,59 $P>0,05$	Death	9
II	2,70±0,21	4,49±0,85	12,51±1,04	Survival	10
	1,38±0,35	5,29±1,64	13,77±3,49	Death	3

TABLE 2. Changes in Plasma PBI Concentration (in μg%) during Experiment (M±m)

	Plasma PBI concentration (in μg%)						Outcome of resuscitation
	Before terminal state	During agony	Recovery period				
			initial state	after 6 h	after 12 h	after 24 h	
I	1,21±0,21 (32)	1,04±0,46 (17)	1,16±0,16 (20)	0,66±0,23 (11)	0,72±0,38 (9)	0,95±0,43 (10)	Survival
				1,82±0,28 (8) $P<0,01$	1,15±0,35 (5)	—	Death
II		—	1,49±0,5 (12)	0,85±0,37 (10)	—	—	Survival
				3,2±0,63 (3) $P<0,01$	—	—	Death

[3, 4, 5, 8, 9, 10] indicating inhibition of thyroid function and stimulation of absorption of thyroid hormones by the tissues in states of stress (hypoxia, shock, surgical operations, etc.). On the other hand, the thyroid may not exhibit this response, or it may be activated in the case of exposure to a combination of extreme stimuli, during stress in decorticated animals and against the background of an excessive response of the pituitary – adrenal system. The special role of this last factor in the activation of thyroid function in the present experiments is indicated by results which have been published [2] showing a prolonged sympathico-adrenal response and activation of the adrenal cortex in the case of severe neurological disturbances during the postresuscitation period.

A link is thus observed between the character of changes in thyroid function and the severity of the course of recovery after resuscitation and its outcome.

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